Nasal Resistance in Obstructive Sleep Apnea*

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Background: Acute increases in nasal resistance are known to induce upper airway occlusion in predisposed subjects. With the limited efficacy of nasal surgery alone in the treatment of obstructive sleep apnea (OSA), the relevance of chronically increased nasal resistance in the pathophysiologic features of OSA remains undetermined.

Methods: Seventy-one patients with OSA (apnea + hypopnea index >15 [AHI]) and 70 antisocial snorers (ASS [AHI <15]) referred for routine assessment of sleep-disordered breathing had concomitant measurement of combined (CNR) and highest unilateral (HUNR) nasal resistance by anterior rhinomanometry.

Results: Nine individuals (five of 71 in the OSA group and 4 of 70 in the ASS group, 0.5<p<0.75) had an abnormally elevated CNR. The HUNR was increased in 21 individuals (11 of 70 in the ASS group and 10 of 71 in the OSA group, 0.25<p<0.5). There was no significant difference between CNR in OSA (1.5[0.5]) (mean [SE]) and ASS (1.6 [0.2]) groups. No correlation was found between CNR and the AHI in OSA or in ASS. The Mean HUNR in the OSA group was 5.5 (0.9) (mean [SE]) and in ASS was 5.3 (0.6) which were not significantly different (p=0.89). The HUNR also did not correlate with the AHI in either OSA or ASS.

Conclusion: Chronic changes in nasal resistance are not a significant risk factor for the development or severity of OSA.


AHI=apnea hypopnea index; ASS=antisocial snorers; CNR=combined nasal resistance; HUNR=highest unilateral nasal resistance; OSA=obstructive sleep apnea.

The level of obstruction and the pathogenesis of obstructive sleep apnea (OSA) have been extensively studied in the past.1-3 Studies show that upper airway caliper or resistance in awake subjects with OSA has no predictive value in determining the site of obstruction during sleep.4 Although acute increases in nasal resistances induce upper airway occlusion in predisposed subjects,5 the relevance of chronically increased nasal resistance in OSA is controversial.6 It has been speculated that increased nasal resistance is associated with increased snoring and apneic events during sleep, but surgical correction of these problems does not resolve OSA.7,8 The aim of the present study was therefore to measure highest unilateral (HUNR) and combined (CNR) nasal resistance to assess its importance in the development and severity of OSA.

METHODS

One hundred forty-one consecutive patients referred to the Sleep Laboratory of the North West Lung Centre, in Manchester, with symptoms suggestive of OSA, had routine concomitant measurement of nasal resistance by active anterior rhinomanometry6 at the time of their first referral. These measurements were made with patients in the erect position. A transparent plastic mask with an inflatable cuff was used. A pressure catheter was attached to the nostrils with adhesive tape. This provided an airtight seal with minimal distortion of the nostrils. The nasopharyngeal pressure and flow were measured with a linear pneumotachograph and plotted on a X-Y recorder (Erich Jaeger, United Kingdom).6 A minimum of three readings with a variability of less than 5 percent were taken. Nasal resistance was calculated at standard pressure (150 pas) and expressed in centimeters of H2O per liter per second.8 The CNR was derived using 1/CNR=1/left nasal resistance +1/right nasal resistance. The patients then underwent polysomnography with EEG, electrooculogram, and electromyogram,11 ECG, finger probe oximetry (Ohmeda Biox 3700E), and respiratory movements recorded as chest and abdominal effort using surface apnea monitors (Densa, United Kingdom). The data were collected via a computerized system (CASS, CNS, United States).

Subsequently, the patients were divided on the basis of the polysomnographic analysis into OSA (apnea hypopnea index [API] >15), n=71, and antisocial snorers (ASS) (AHI <15), n=70. All values unless specified are expressed as mean±SD. The technician analyzing the sleep study was unaware of the nasal resistance measurements.

Statistical analysis was performed using the Statworks package (Macintosh). The HUNR and CNR in the two groups were compared using the Student's t test. Correlations were made with simple linear regression analysis, and comparison of frequencies of abnormal nasal resistance between groups was made with the x² test.

RESULTS

Both groups were primarily male and middle-aged (Table 1). The body mass index was significantly higher in OSA than in the ASS group (p<0.001). The CNR was similar in both groups (p=0.72). Similarly, the HUNR was not significantly different (p=0.89) in both groups. There

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was no correlation between the AHI and CNR (r=0.1; p=0.27) or HUNR (r=0; p=0.27). Nine individuals (4 of 70 in the ASS and 5 of 71 in the OSA group; 0.5<p<0.75, not significant by \( \chi^2 \) test) had a CNR outside our laboratory’s normal range (>3 cm H\(_2\)O/L/s). Twenty-one individuals had an HUNR greater than 8 cm H\(_2\)O/L/s (11 of 70 in the ASS and 10 of 71 in the OSA group; 0.25<p<0.5, not significant), the normal range being 2 to 8 cm H\(_2\)O/L/s.\(^6\) In this subgroup of patients with elevated HUNR, when HUNR was correlated with the AHI, the coefficient of correlation in the OSA group was 0.3, whereas that in the ASS group was 0.1. Four OSA patients with increased HUNR also had an increased CNR, while only one in the ASS group had increased CNR associated with increased HUNR.

The percentage of total sleep time a patient spent on his side with the nostril with the HUNR at the lowest level was defined as ipsilateral percent. There was no significant correlation between the CNR and the ipsilateral percent in the OSA (p=0.84; r=0) or ASS (p=0.5; r=0.1) groups. Similarly, there was no correlation between the HUNR and the ipsilateral percent, in either the OSA (p=0.65; r=0.1) or the ASS (p=0.57; r=0.1) group. No relationship could therefore be demonstrated between the HUNR or CNR and the sleep position.

**Discussion**

The pathophysiology of OSA involves the development of pharyngeal airway narrowing.\(^6\) The pharyngeal airway may behave like a Starling resistor due to the decreased upper airway muscle tone and phasic inspiratory activity during sleep.\(^12\) The collapsibility of the compliant wall is further enhanced by the increased resistive load posed by obesity which alters the physical characteristics of the walls. The subatmospheric pressure generated within the collapsible segment contributes to the inspiratory flow limitation.\(^6\) Maximal flow limitation occurs at the site of the smallest cross-sectional area in the upper airway.\(^13\) Although this mainly involves upper airway resistance as a whole, the contribution of nasal resistance toward this phenomenon remains undetermined. By increasing the upstream resistance, increased nasal resistance might increase the pressure drop across the narrowest segment in the pharynx, contributing to occlusion. This might aggravate the problems of snoring and OSA, for example, with nasal polyps, hypertrophied turbinates, adenoid hypertrophy, and nasal congestion.\(^14\) But surgical correction does not resolve OSA.\(^7\)\(^8\)

Experimentally induced acute nasal occlusion affects breathing during sleep and sleep quality possibly due to an inability to adapt the respiratory pattern to this sudden change.\(^5\)\(^15\)\(^17\) Although, a previous study showed an increase in the AHI from a mean of 0.7 to 1.7 following a doubling of nasal resistance in allergic rhinitis,\(^16\) the increase in the AHI was clinically insignificant. The nasal resistance measurements in our patients were made in the chronic stable state, when compensatory changes would presumably have occurred. These measurements would therefore seem more relevant in determining the clinical significance of nasal resistance in the OSA or ASS groups. In the present study, an elevated CNR only occurred in 7 percent of OSA and in 5.7 percent of the ASS group, which is clinically insignificant for the whole group. Nasal resistance did not correlate with the severity of OSA. The absence of elevated CNR or HUNR in a significant proportion of our patients in the OSA and ASS groups could imply that either daytime measurement of nasal resistance may not reflect the pressure flow changes occurring at night or that nasal resistance is unimportant in relation to total upper airway resistance at night.

Nasal resistance is subject to diurnal and postural variations. It is maximal at night and in the early morning.\(^19\)\(^21\) It is increased in the supine and lateral recumbent positions, with the resistance on the dependent side being higher than that on the superior side.\(^22\)\(^24\) Our patients, however, had HUNR measured in the erect position. We considered the possibility that patients with chronically increased HUNR from either group may sleep predominantly in the lateral recumbent position with the blocked nostril at the lowest level. This bias, however, could not be demonstrated in either group. Although diurnal variation of nasal resistance has not been investigated in OSA, it could be argued as a factor determining pharyngeal collapse at a critical transmural pressure. However, nasal resistance only changes minimally in transition from wakefulness to sleep in comparison with the large and variable increase in resistance of the supralaryngeal airway.\(^13\) In addition, no direct correlation could be demonstrated between the severity of nasal obstruction and the severity of OSA, when Olsen and...
Kern\textsuperscript{7} studied acute nasal obstruction during sleep. Patients with OSA may have symptomatic improvement in daytime hypersonmolence after nasal surgery, but it has never been shown convincingly to resolve the obstructive apneas. Tolerance to nasal constant positive airway pressure also improves after nasal surgery probably due to relief of nasal airflow impediment.\textsuperscript{5}

In conclusion, the present study did not find daytime nasal resistance to be a risk factor for OSA. Nasal resistance as a major determinant of inspiratory flow limitation in OSA would seem unlikely in light of the present observations. Nasal surgery for improving resistance in OSA is only indicated for relief of symptoms of daytime nasal obstruction.

REFERENCES

5 Suratt PM, Turner BL, Wilhoit SC. Effect of intranasal obstruction on breathing during sleep. Chest 1986; 90:324-29
6 Kuna ST, Sant 'Ambrogio G. Pathophysiology of upper airway closure during sleep. JAMA 1991; 266:1384-89